A Post Traumatic Osteoarthritis Model for Screening Therapeutics and Related Mechanically-Activated Drug Delivery

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Where: B2.74, Trinity Biomedical Sciences Institute

Cartilage regeneration and repair are perplexing clinical problems with numerous approaches being studied using tissue engineering and novel therapeutics. It is commonly accepted that one route cause or initiator of osteoarthritis is preceding joint trauma. While there are a number of in vitro models of post-traumatic osteoarthritis (PTOA) have been developed to study the effect of mechanical overload on the processes that regulate cartilage degeneration. While such frameworks are critical for the identification therapeutic targets, existing technologies are limited in their throughput capacity. This presentation will describe one such new approach using a validated a test platform for high-throughput mechanical injury incorporating reproducible engineered cartilage we refer to as cartilage tissue analogs (CTA). Studies will be described demonstrating the response of the CTA to a single compressive injury and further will discuss the screening of putative PTOA therapeutic compounds and small molecules.

Since the joint presents a unique and challenging environment with specialized matrix and mechanical environment, drug delivery poses special opportunities and difficulties. The second part of the talk will discuss a new approach to deliver therapeutics to improve regeneration using mechanically activated microcapsules. The second part of the presentation will describe a novel and tunable approach to drug delivery, their rupture profiles and will demonstrate the effectiveness of growth factor delivery to cells using microcapsules.

The studies described in this presentation establishes a high throughput tool for the discovery of mechanisms governing cartilage injury, as well as a screening platform for the identification of new molecules for the treatment of PTOA and moreover, new information describing a novel drug delivery system taking advantage of the mechanically charged environment of the joint. Ultimately the studies presented here will hopefully show both a valid approach that will identify compounds active at promoting cartilage health and regeneration and begin to address the unsolved clinical issue of cartilage degeneration and OA.

George R. Dodge, Ph.D., is an established investigator with a career long commitment to the field of cartilage biology and therapeutic discovery research related to osteoarthritis. Dodge is currently Associate Professor of Orthopaedic Surgery at University of Pennsylvania in the Department of Orthopaedic Surgery, and the Department of Otorhinolaryngology. Dodge is Director of the Translational Musculoskeletal Research Center at the Philadelphia Department of Veterans Affairs Medical Center. He also directs the newly formed Otorhinolaryngology Translational Regenerative Research Program. Dodge began his academic training at Asbury College, followed by earning a bachelor’s degree from the State University of New York in Biology and Public Health before obtaining a Ph.D. at McGill with A. Robin Poole at the Shriner’s Hospital for Children. Upon return to the US he completed a fellowship and faculty positions at Thomas Jefferson University in Philadelphia. Dodge has spent more than 25 years focused on research in the area of arthritis and in particularly related to understanding osteoarthritis and has developed novel approaches to identify new therapies. Currently Dodge is at the University of Pennsylvania having research laboratories in the McKay Orthopaedic Research Laboratory, Department of Orthopaedic Surgery and at the VA Medical Center. His research currently focuses on musculoskeletal injuries and exploring how to both repair cartilage injuries through tissue engineering as well as exploring new therapeutics. The Dodge research approach incorporates a multidisciplinary approach in ongoing research involving both structure and function and ultimately identifying ways to alleviate pain and loss of function for patients.